WHAT IS CLAIMED IS:

1. Anti-CEA/NCA antibodies, which comprise antibodies raised against subdomains of CEA/NCA involved in differentiation-blocking activity associated with tumorigenicity, wherein said subdomains are selected from the group consisting of sequences G_{30}YSWYK (SEQ ID NO:1), N_{42}RQII (SEQ ID NO:2), Q_{80}ND and other sequences in the N terminal 107 amino acid domain, and sequences in the internal A3B3 178 amino acid domain of CEA.

2. The antibodies of claim 1, wherein said antibodies release CEA/NCA-imposed differentiation block in CEA/NCA-producing tumors and their metastases in a cancer patient.

3. Peptides and peptide-derived mimetics, which comprises peptide and peptide-derived mimetics interacting with subdomains of CEA/NCA involved in the differentiation-blocking activity associated with malignant tumors, wherein said subdomains are selected from the group consisting of sequences G_{30}YSWYK (SEQ ID NO:1), N_{42}RQII (SEQ ID NO:2), Q_{80}ND and other sequences in the N-terminal 107 amino acid domain, and sequences in the internal A3B3 178 amino acid domain of CEA.

4. An inhibiting CEA/NCA sequence, which comprises antisense cDNA, oligonucleotide or ribozyme sequences which hybridize to at least one domain of CEA/NCA selected from the group consisting of mRNA sequences of CEA and NCA which reduces expression of CEA/NCA in tumors and metastases when administered to a cancer patient.

5. The inhibiting CEA/NCA sequence of claim 1, wherein said sequence is an antisense cDNA, an antisense oligonucleotide or an antisense ribozyme.

6. A shankless anchor, which comprises a GPI anchor of CEA without the external domains, wherein said GPI anchor interferes with downstream targets of endogenous CEA/NCA molecules to inhibit differentiation-blocking activity of endogenous CEA/NCA molecules when administered to a cancer patient.

7. A method to restore endogenous integrin function, which comprises the steps of:

a) administration of monoclonal antibodies that reverse EA/NCA-induced changes in integrin function; and

b) administration of peptides/mimetics that mimics the effect of the mAbs;

thereby inhibiting differentiation-blocking activity of the endogenous CEA/NCA molecules.

8. The method of claim 7, wherein said integrin function includes integrins $\alpha_5\beta_1$ and $\alpha_v\beta_3$.

9. A drug screen assay utilizing CEA/NCA-expressing transfectants of rat L6 myoblasts to determine pharmaceutical agents which are capable of inhibiting signaling process required for differentiation-blocking activity of the endogenous CEA/NCA molecules, which comprises the steps of:

a) screening for agents capable of releasing myogenic differentiation block in rat L6 cells expressing CEA/NCA; and

b) screening for agents capable of restoring normal cellular and tissue architecture to human Caco-2

colonocytes aberrantly expressing high levels of CEA/NCA.

10. The use of the anti-CEA/NCA antibodies of claims 1 and 2, the peptides and peptide-derived mimetics of claim 3, the inhibiting CEA/NCA sequence of claims 4 and 5 or the shankless anchor of claim 6, to enhance efficacy of other anti-cancer treatment by increasing differentiation status of a tumor and by enhancing bystander effect; whereby more differentiated tumor cells cause more adjacent autonomous tumor cells to behave more as non-malignant or normal cells.

11. The use of the anti-CEA/NCA antibodies of claims 1 and 2, the peptides and peptide-derived mimetics of claim 3, the inhibiting CEA/NCA sequence of claims 4 and 5 or the shankless anchor of claim 6, to restore anoikis/apoptosis to levels of non-malignant or normal cells, thereby increasing efficacy of all other cyto-toxic chemotherapeutic drugs which depend on apoptosis for killing cells.

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